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Audio-vestibular signs in rheumatoid arthritis

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ABSTRACT

Aims: The purpose of this study is to investigate the audio vestibular signs in patients with rheumatoid arthritis (RA).

Methods: In this cross sectional comparative research, the study group included 30 RA patients and 30 control group patients who had no ear complaints. All patients had audio vestibular evaluations that include pure tone audiometry, tympanometry, acoustic reflexes, otoacoustic emissions (OAE) and vestibular evoked myogenic potential (VEMP).

Results: In RA group pure tone audiometry; air conduction at 500, 1000, 4000 Hz frequencies, bone conduction at 500, 4000 Hz frequencies were having more hearing loss than the control group. Tympanometry showed that pressure and the gradient were increased in RA group compared with control group. In acoustic reflex test; the amplitude values were less than the control group at 500, 1000, 4000 Hz frequencies in RA group's contralateral ear. When the RA group compared with control group in OAE testing, amplitude values of TEOAE was decreased in RA group at 1000, 2000 Hz frequencies, and at 1000 Hz frequency, the amplitude was decreased in DPOAE. In VEMP; N1 latency amplitude was longer than the control group and P1N1 amplitude was shorter than the control group.

Conclusion: When the systemic effects and etiopathogenesis of RA was thought; it was concluded that and audio vestibular system may be affected by this way

Keywords: audio-vestibular, rheumatoid arthritis, vestibular evoked myogenic potentials

INTRODUCTION

Rheumatoid arthritis (RA) is a connective tissue disease characterized by chronic inflammation of the synovial joints.[1] Although its etiology is not known exactly, it is a multisystemic disease, and there have been studies in recent years suggesting that it may cause hearing impairment as well as its neurological, temporomandibular, and laryngeal reflections. It is argued that autoimmunity plays a role in the etiology.[2] It is thought that the target of this autoimmunity may be type II collagen or a glycoprotein in articular cartilage.[2] In the cochlea, the basilar membrane separating the scala media and scala tympani from each other is connective tissue and is rich in glycoprotein and fibronectin.[2] The lamina spiralis also contains type II collagen, the limbal layer, and the tectorial membrane covering the spiral limbus cells, which are attached to the inner edge of the ossea.[2] It can be thought that the autoimmunity that develops against the collagen and glycoprotein structure in RA may also develop against the glycoprotein and collagen structure present in the inner ear.[2,3]

METHODS

Study Population and Design

This study was approved by the Ethics Committee of Kırıkkale University, Faculty of Medicine (Date 21.02.2011, Decision No: 2011-0021). Informed consent was obtained from all participants included in the study. All procedures were carried out in accordance with the ethical rules and the principles of the Declaration of Helsinki. This study is a cross-sectional comparative study. Thirty patients were included in study (6 males, 24 females)who were diagnosed as having RA after anamnesis, clinical examinations, and laboratory tests in accordance with the ARC 1987 criteria, by the Department of Physical Medicine and Rehabilitation in Kırıkkale University.[4] The control group consisted of 30 patients (16 men and 14 women) who applied to the Ear, Nose, and Throat (ENT) Department for reasons other than ear complaints. Otoscopic examination of all control group was normal.

Methods for Evaluation

Pure-tone audiometry, impedancemetric measurements (tympanometry and acoustic reflex), otoacoustic emission,

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and vestibular evoked myogenic potential (VEMP) tests were applied to the patients. In pure-tone audiometry, Losses greater than 20 dB relative to the pure tone average were considered hearing loss. Patients with air and bone conduction gaps of more than 10 dB were considered to have CHL. Patients whose pure tone averages were higher than 20 dB and the air-bone gap was less than 10 dB were considered SNHL. Patients with a difference of more than 10 dB were considered to have mixed hearing loss and pure tone average (PTA) calculated by taking the average of the values obtained at 500, 1000, and 2000 Hz frequencies in the air and bone threshold. Tympanometry and stapes reflex (acoustic reflex) were measured in impedancemetry. Pressure and gradient values were measured in the obtained tympanogram curve. In acoustic reflex measurement, the stapes reflexes of the patients were measured ipsilaterally and contralaterally at frequencies of 500, 1000, 2000, and 4000 Hz, and the reflex was recorded as "yes" or "no". In the otoacoustic emission, the amplitude values obtained in the transient evoked otoacoustic emission (TEOAE) and distortion product otoacoustic emissions (DPOAE) were recorded. Afterwards, a cervical VEMP (c-VEMP) test was performed on the patients, and VEMP values (P1 latency (milliseconds, ms), N1 latency (ms), P1N1 latency (ms), and P1N1 amplitude (microvolts, V) were recorded.

Statistical Analysis

A statistical analysis was made using the SPSS 23 package statistics program. The means of the parameters that did not fit the normal distribution were compared with the Mann-Whitney-U test. The comparison of the groups was made using the chi-square test. p value below 0.05 was considered significant.

RESULTS

A total of 30 patients (7 men and 23 women) from the RA patient group were included in this study. The ages of the patients were between 20 and 70 years, and the mean age was 49.76 years. The control group of the study consisted of 14 men and 16 women, for a total of 30 patients that ages were between 36 and 65, with a mean age of 44.83 years (**Table 1**). In the RA group, CHL was detected in 8 ears (13.3%) and SNHL in 15 ears (25%). There was no mixed-type hearing loss in any of the patients, and hearing in 37 ears was evaluated as normal. SNHL was detected in 7 ears (11.6%) in the control group. The evaluated normal ears were 53. Hearing loss was more common in the RA group at 500, 1000, and 4000 Hz between the airway hearing thresholds of the control and RA groups ($p<0.05$). Hearing loss was found to be higher in the RA group for the bone conduction threshold at 500 and 4000 Hz frequencies between the bone conduction hearing thresholds in the control and RA groups ($p<0.05$). In the control and RA groups, both parameters were higher in the patient group, between pressure and gradient values. In acoustic reflex measurements no statistically significant results were found in the ipsilateral and contralateral ears between the control and RA groups ($p<0.05$). In otoacoustic emission measurement, control and RA TEOAE amplitude values at 1000–4000 Hz frequencies were found to be lower in the RA group at 1000 and 2000 Hz frequencies ($p<0.05$). In DPOAE amplitude values, the amplitude values were found to be lower in the RA group at a frequency of 1000 Hz ($p< 0.05$). P1 latency (ms), N1 latency (ms), P1N1 latency (ms), and P1N1 amplitude (mV) were evaluated in the VEMP test. Between the control and RA groups, N1 latency was found to be prolonged in the RA group compared to the control group, and the P1N1 amplitude was found to be decreased ($p< 0.05$). The results of the study are shown in **Table 2**.

Table 1. Demographic data

	Rheumatoid arthritis group n=30	Control group n=30	All n=60
Age	49.76±11.30	44.83±7.40	47.30±9.68
Gender			
Male	7 (23)	14 (47)	21 (35)
Female	23 (77)	16 (53)	39 (65)

(%) Column Percentages

Table 2. Comparison of Rheumatoid Arthritis (RA) and control group (n=60)

		Mean±sd	Min-max	P
Air Conduction Results (pure tone audiometry)				
500 Hz (dB)	Control RA	13.25±7.41 21.00±7.90	5.00-40.00 5.00-40.00	0.001
1000 Hz(dB)	Control RA	13.58±8.54 17.50±8.51	5.00-50.00 0.00-40.00	0.01
2000 Hz (dB)	Control RA	14.00±8.82 17.16±9.26	0.00-50.00 5.00-40.00	0.06
4000 Hz (dB)	Control RA	17.16±10.26 21.91±12.31	5.00-50.00 5.00-70.00	0.02
TEOAE Results				
1000 Hz (dB)	Control RA	3.53±5.20 1.34±2.97	0.00-20.50 0.00-13.90	0.005
1500 Hz (dB)	Control RA	4.65±5.25 3.98±5.71	0.00-19.20 0.00-19.60	0.51
2000 Hz (dB)	Control RA	6.84±4.63 4.26±5.29	0.00-19.40 0.00-19.60	0.004
3000 Hz (dB)	Control RA	4.46±4.25 3.81±4.82	0.00-13.30 0.00-19.40	0.43
4000 Hz (dB)	Control RA	1.83±3.75 2.59±4.27	0.00-11.60 0.00-14.90	0.3
VEMP Results				
P1 latency (ms)	Control RA	14.25±2.88 16.96±11.29	8.33-24.67 0.00-48.00	0.054
N1 latency (ms)	Control RA	20.89±4.08 23.93±14.44	12.33-40.33 0.00-55.33	0.03
P1N1 latency (ms)	Control RA	6.67±2.67 6.97±4.71	3.33-15.67 0.00-20.67	0.66
P1N1 amplitude (μV)	Control RA	6.09±2.35 4.95±3.44	2.20-12.10 0.00-13.00	0.02

TEOAE (Trans Evoked Otoacoustic Emissions), VEMP (Vestibular Evoked Myogenic Potentials)

DISCUSSION

Baradanafar et al.[5] performed PTA, tympanometry, and acoustic reflex tests in patients with RA in 2010. Significant difference were obtained at 8000 Hz in PTA. It has been suggested that the current SNHL may be an extra-articular reflection of RA and may be due to drug use or ototoxicity. It has been claimed that increased laxity in the middle ear transduction mechanism due to damage to the joint fluid between the middle ear ossicles may be a cause of CHL.[5] It has been suggested that mixed hearing loss may be related to the multifocal effect of the audiological system.[5] There are also studies where it is claimed that SNHL may be due to 8th nerve damage or vasonervosum arteritis.[6] Raut et al.[7] on the other hand, claimed that SNHL could be due to immune complex-associated vasculitis occurring in the inner ear, antibodies developing against the inner ear, neuritis, or ototoxic effects of drugs. They suggested that

CHL may be due to joint destruction between the ossicular chain. There are other studies suggesting that SNHL may be due to vasculitis or neuritis in parallel with Raut et al.[8-10] There are many studies showing that SNHL is mostly detected in RA.[5-7,9]

In the studies performed, SNHL was reported in 27–41% of patients with RA in general; CHL was detected at a rate of 17.4%.[6,7,10] In current study, hearing loss was found in approximately 50% of the cases. 13.3% of this hearing loss was evaluated as CHL and 25% as SNHL.

The involvement of middle ear ossicles in RA was first suggested by Copeman.[11] Copeman found CHL in 3 RA patients with hearing loss who had no pathological physical examination findings related to the ear. He emphasized that this situation may be related to the involvement of middle ear ossicles by RA. Heyworth et al.[10] found sensorineural hearing loss in 27% of patients with RA in their study; they could not find any evidence that RA affects the ossicular chain. Rosenberg[12] and Moffat[13] suggested that CHL is due to a decrease in thickness in the tympano-ossicular system, while Reiter[14] suggested that the rigidity of the middle ear transduction mechanism increases. In current study, the majority of cases were SNHL. From this point of view, it has been concluded that the cochlear structures are more affected than the middle ear ossicles.

Ozcan et al.[15] evaluated the tympanometry values as abnormal, although not significant. Halligan et al.[16] found no difference between the tympanometry results of the RA and control groups. Coletti et al.[17] found a relationship between abnormal resonance values in tympanometry and more aggressive RA, suggested that RA may have affected the incudostapedial and incudomalleolar joints, which are diarthrosis joints. In study, pressure and gradient values were found to be higher in the RA group. This situation may be due to the extra-articular involvement of the diarthrosis joints in the middle ear, in parallel with the comments of Ozcan et al.[15] There are studies on how much the auditory system is affected in patients with RA, but there are very few studies on how much the vestibular system is affected by these multisystemic diseases. Heydari et al.[18] performed the VEMP test in patients with RA and found the P13 latency to be higher in the patient group. No difference was found between amplitudes. Ferrara et al.[19] found vestibular dysfunction in some RA patients. Kakani et al.[20] obtained abnormal caloric test results or abnormal saccadic eye movements in patients with RA. King et al.[21] suggested that vestibular involvement in RA may cause vision loss due to vestibulo-ocular and optokinetic reflex involvement and postural instability due to vestibulospinal reflex dysfunction. In our study, N1 latency increased and P1N1 amplitude decreased in patients with RA in the c-VEMP test. This suggested an abnormality in the vestibulosaccular pathway.

Limitations of This Study

This is because it was done on a small group of patients. Our measurements reflect only a cross-section of time. This relationship could have been better studied in a cohort study with repeated measurements.

CONCLUSION

RA is a disease that still has many unknown factors in its etiopathogenesis and is thought to be caused by genetic, environmental, hereditary, and autoimmune causes.. It was concluded that the entire audiovestibular system, especially the cochlear and vestibular end organs, may be affected in RA.

ETHICAL DECLARATIONS

Ethics Committee Approval: The study was carried out with the permission of Kırıkkale University, Faculty of Medicine Ethics Committee (Date 21.02.2011, Decision No: 2011-0021).

Informed Consent: All patients signed the free and informed consent form.

Referee Evaluation Process: Externally peer-reviewed.

Conflict of Interest Statement: The authors have no conflicts of interest to declare.

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Author Contributions: All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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